

# PostScript

## CORRESPONDENCE

### The aetiology of flaccid paralysis in West Nile virus infection

We read with interest the recent article by Park *et al.*<sup>1</sup> describing a syndrome of acute anterior radiculitis associated with West Nile virus (WNV) infection. Although admittedly there is still much to learn about the clinical spectrum of disease associated with WNV, we were troubled by several of the assertions raised by the article, and the conclusions drawn.

Recent evidence has suggested that the majority of patients developing acute asymmetrical weakness in the setting of WNV infection suffer from damage to spinal anterior horn cells, resulting in a poliomyelitis-like syndrome.<sup>2-5</sup> This has been supported by electrodiagnostic data<sup>2-5</sup> and by pathology demonstrating the destruction of anterior spinal grey matter.<sup>6-9</sup> Park *et al.* assert that "the mechanism of weakness associated with WNV infection continues to be unclear", and they subsequently "propose an alternate explanation for the associated weakness". This alternative explanation of "acute anterior radiculitis" is based on MRI findings that showed intradural lumbosacral nerve root enhancement in a patient with unilateral leg weakness.

However, the authors do not describe MRI findings in the anterior spinal cord, and the MRI images provided are at the L1-L2 and L2-L3 levels, which lie caudal to the cord segments giving rise to lumbar roots; MRI of the thoracic spine is needed to adequately visualise lumbar cord segments. This is an important omission, since it is unclear whether the authors visualised the anterior lumbar cord before proposing an alternative explanation for WNV associated weakness. In addition, the authors' contention that their case displayed ventral nerve root involvement can be challenged, since it is difficult to distinguish intradural ventral nerve root enhancement from posterior root enhancement. Furthermore, nerve root enhancement in and of itself is a relatively non-specific finding which may be seen with meningeal inflammation in general, and may not have a clinical correlate. Accordingly, the authors' argument that the MRI findings call into question the pathophysiology of weakness, and provide evidence for an anterior radiculopathy, rather than a poliomyelitis, as the aetiology of weakness, is speculative.

It is true that definitive MRI changes have thus far been absent in many cases of WNV poliomyelitis, although clear documentation of such changes has been reported.<sup>9</sup> However, neuroimaging data in persons with poliomyelitis due to wild-type poliovirus or other neurotropic viruses has not been consistently gathered, and the incidence of such MRI changes is unknown. It is likely that specific MRI changes may be missed because of variations in the stage of disease at the time of imaging. Finally, the transient reversible muscle weakness seen in this case differs clinically from that in individuals with WNV poliomyelitis-like syndrome, who develop chronic profound weakness.<sup>10</sup>

It is clear that WNV infection may be associated with a myriad of clinical and pathophysiological features, and transient

weakness due to anterior radiculitis certainly may be among those features. However, generalisation based on one rather atypical case, and invoked to propose an alternative explanation for the flaccid paralysis seen in WNV infection, is problematic. It is our opinion that poliomyelitis clearly has been established as the aetiology of most cases of acute, asymmetrical paralysis seen in WNV infection.

The article by Park *et al.* raises an important issue, however, about the utility of MRI in the diagnosis of WNV; such questions about MRI findings both in WNV poliomyelitis-like syndrome and in WNV encephalitis may be addressed by serial MRI assessments of patients during and following acute illness.

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## Authors' reply

Our understanding of the West Nile Virus (WNV) infection and its neurological manifestations has rapidly expanded in recent months. The comments submitted by Dr Leis and colleagues raise concerns regarding the case we presented. The data they cite as contrary evidence to our conclusions were published after the submission of our report (accepted for publication 10 February 2003). Since that time, pathological evidence in human cases has emerged that implicates the spinal cord anterior horn cell in the pathogenesis of WNV associated flaccid

paralysis. Nonetheless, the clinical, electrodiagnostic, and MRI findings presented in our case are still valid.

In our specific case, the patient clearly exhibited signs and symptoms of an acute lower extremity motor paralysis that was supported by electrodiagnostic studies. It is important to reiterate here that such studies are incapable of distinguishing whether the pathology is located in the anterior horn, ventral root, or motor axon. As such, the conclusions and pathological data that preceded our report were potentially premature in implicating only the anterior horn cell, despite the clinical presentation of a poliomyelitis-like syndrome.

The MRI study we presented showed enhancement of the ventral nerve roots. In the published image no signal change was seen within the adjacent spinal cord itself. Although the nerve roots may enhance with a non-specific meningeal process, the area of signal abnormality in our patient correlated well with the clinical and electrodiagnostic findings. In addition, signal change was not seen in other areas of the MRI scan. Previous MRI studies of poliovirus infection, as well as non-poliovirus infection that involved the anterior horn cell, demonstrated signal changes and enhancement in the region of the anterior horn cell within the spinal cord.<sup>1-4</sup> As a result, we concluded that because no such enhancement was seen in the anterior horn cell region, the suspected pathogenesis in our case study may have extended from the anterior horn to include the ventral nerve root.

It has now been demonstrated by pathological studies that the anterior horn cell is affected in cases of flaccid paralysis caused by the WNV; however, lymphocytic infiltration of the nerve root was also seen.<sup>5</sup> At this time, we conclude that perhaps the nerve root, in addition to, or independent of, the anterior horn cell, can also be involved in acute flaccid monoparesis caused by the WNV.

As our understanding of WNV infection and its neurological manifestations continues to evolve, it remains important to consider varied presentations of the disease, even if at times they appear contradictory. Unfortunately, MRI is an insensitive tool for assessing WNV infection. It is our hope that future prospective and pathological studies will continue to advance our understanding of the pathophysiological mechanisms that underlie the WNV infection.

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## Spontaneous retinal venous pulsations can be present with a swollen optic disc

I read with interest the article "Spontaneous retinal venous pulsation: aetiology and significance" by Jacks and Miller.<sup>1</sup> Their explanation for these pulsations is essentially no different from that put forward by Levine in 1998.<sup>2</sup> They then go on to discuss the clinical importance of spontaneous retinal venous pulsations (SVPs). They refer to the finding of Levin that the presence of SVPs is an indication of an intracranial pressure below 190 mm H<sub>2</sub>O.<sup>3</sup> However, they conclude without justification that "presence of SVPs allows the examiner to conclude that the patient does not have optic disc swelling..."

We cannot conclude that because an individual has SVPs there is no true disc swelling. Shortly after their article was published, a 53 year old man was referred to us by his optometrist with "raised discs". On examination, he was found to have markedly elevated optic discs, with an SVP in the left eye. After a normal head CT scan, the patient had a lumbar puncture with an opening pressure of 400 mm H<sub>2</sub>O, leading to a diagnosis of papilloedema secondary to idiopathic intracranial hypertension. If we had concluded that the discs were not in fact truly swollen (pseudopapilloedema), we would have had no justification for performing these investigations which lead to treatment, and the patient would have been much more likely to suffer irreversible visual loss. Persons with disc swelling due to ischaemic or inflammatory causes may also have SVPs in the affected eye, although less often than in unaffected eyes.<sup>4</sup>

The presence of SVPs can be used to provide an upper limit for a patient's CSF pressure, but says nothing about whether or not the disc is swollen. The finding of an SVP can indicate whether papilloedema is likely, but not whether it is actually present, as illustrated by our case. Perhaps the best message on this topic derives from an authoritative neuro-ophthalmic text: "The observation of spontaneous venous pulsations indicates only that ICP is below 200 mm of water at that time, not that the patient does not have papilloedema."<sup>5</sup>

Jacks and Miller have provided a review based mostly on two original articles, one of which quite adequately described the clinical significance of SVPs. They have then in our opinion drawn unfounded, incorrect, and inevitably harmful conclusions.

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## Author's reply

We thank McKee and Ahad for their letter in which they question our statement that "the presence of spontaneous venous pulsations (SVPs) allows the examiner to conclude that the patient does not have optic disc swelling". Although we believe this statement to be generally true, and agree with McCulley *et al*<sup>1</sup> that most discs with optic disc swelling do not show SVPs, persons with mild papilloedema, particularly individuals with pseudotumour cerebri, have significant fluctuations in intracranial pressure (ICP). Such people may indeed show SVPs during the period throughout which their ICP is normal. Thus, the decision as to whether or not an elevated disc is truly swollen should never be made entirely on the presence or absence of SVPs, but on the entire clinical picture.

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## BOOK REVIEWS

### Fetal and neonatal brain injury: mechanisms, management and the risks of practice

Edited by David K Stevenson, William E Benitz, and Philip Sunshine. Published by Cambridge University Press, Cambridge, 2003, pp 886, £140.00 (hardback). ISBN 0-521-80691-7

This book is now in its third edition and is subdivided into six parts that together form a comprehensive review of the aetiology, pathogenesis, and management of the brain injured neonate. The text covers epidemiology; pathophysiology and pathogenesis; pregnancy; complications of labour and delivery; diagnosis of asphyxia; specific conditions associated with fetal and neonatal brain injury; and assessment and management.

The editors have worked hard to "cross-link the basic science with the bedside needs" and have produced a text with clear explanations of the complex issues surrounding the management of the brain injured neonate. They combine a broad vision with attention to detail to produce an extremely useful text.

There is due emphasis given to current issues, such as the role of antenatal infection

in causing cerebral injury and hypothermic neural rescue, and also an eye to the future and issues such as that on near infrared imaging. There is a useful review of the potential for neuroprotective therapy, and up to date contributions on all the standard issues such as long term outcome, treatment of seizures, and drug misuse.

How does the book compare with its competitors? The standard text for most workers is probably Jo Volpe's magisterial single author textbook, and in comparison to this the new volume fares well. There is less basic science—particularly in neuroanatomy and cell biology—but there is a wider clinical scope. I shall keep both volumes on my shelves and will expect to find complementary information in both on any given topic.

**D Edwards**

### Vascular cognitive impairment: preventable dementia

Edited by John V Bowler and Vladimir Hachinski. Published by Oxford University Press, Oxford, 2003, pp 337, £79.50 (hardback). ISBN 0-19-263267-1

This book is an authoritative account of vascular cognitive impairment written by a host of international figures in the field of cerebrovascular disease. The title, *Vascular cognitive impairment*, is significant. The editors regard the more widely used term vascular dementia as having outlived its usefulness. The latter presupposes problems in memory, which are not invariably present, and it defines people relatively late in the course of disease, preventing early diagnosis and treatment. The editors propose adoption of the concept of vascular cognitive impairment and argue for a wholesale revision of current diagnostic criteria.

Despite the title, most of the book is devoted to vascular dementia and its causes and consequences. This emphasis reflects the fact that the chapters, which include the themes of subtypes, cognitive assessment, neuroimaging, histopathology, genetics, and treatment, are predominantly reviews or meta-analyses of published literature. A feature that consistently emerges is the clinical and pathogenic heterogeneity of vascular dementia. Moreover, the reader becomes aware of the obscuration that arises from treating vascular dementia as a uniform entity. For example, whereas cognitive studies X and Y reveal better performance on tests A and B in patients with vascular dementia than patients with Alzheimer's disease, study Z shows the reverse finding. The group data obstruct identification of distinct profiles of impairment relevant to individual patients. It becomes evident why a radical overhaul of thinking about vascular cognitive impairment is required.

My quibble is that like many multi-author texts there is information overlap across chapters. There are also occasional errors overlooked at the proof reading stage. However, in general the book provides a useful, state of the art guide to vascular cognitive impairment. Particularly enjoyable are the editors' lucidly written introductory and concluding chapters, which have a strong personal flavour, and a sense of mission.

**J Snowden**